Osteopathic Manipulative Treatment

Novel Application to Dermatological Disease

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ABSTRACT

Dermatological diseases, such as dysesthesia syndromes, stasis dermatoses, and hyperhidrosis are difficult to treat due to their complex etiologies. Current theories suggest these diseases are caused by physiological imbalances, such as nerve impingement, localized tissue congestion, and impaired autonomic regulation. Osteopathic manipulative therapy targets these physiological dysfunctions and may serve as a beneficial therapeutic option. Osteopathic manipulative therapy techniques include high velocity low amplitude, muscle energy, counterstrain, myofascial release, craniosacral, and lymphatic drainage. An osteopathic manipulative therapy technique is chosen based on its physiological target for a particular disease. Osteopathic manipulative therapy may be useful alone or in combination with standard therapeutic options. However, due to the lack of standardized trials supporting the efficacy of osteopathic manipulative therapy treatment for dermatological disease, randomized, well-controlled studies are necessary to confirm its therapeutic value. (*J Clin Aesthet Dermatol.* 2012;5(10):24–32.)

Dysesthesia syndromes, stasis dermatoses, and hyperhidrosis often present as therapeutic challenges. Often, these conditions are treated with many different classes of topical and systemic agents with variable success. Another form of treatment, known as osteopathic manipulative treatment (OMT), may be of benefit. OMT is a part of the practice of osteopathic medicine that is based on the following four principles:[1] 1) the person is a unit composed of body, mind, and spirit; 2) the body is capable of homeostasis, self-healing, and health maintenance; 3) structure and function are interrelated; and 4) rational treatment is based on an understanding of the above principles. By combining a thorough understanding of anatomy with the pathophysiology of disease, OMT works to restore normal physiological function. The goal of OMT is to treat somatic dysfunction. The *Glossary of Osteopathic Medicine* defines somatic dysfunction as "the impaired or altered function of related components of the somatic system: skeletal, arthroidal, and myofascial structures and their related vascular, lymphatic, and neural elements."[2] Osteopathic physicians use different OMT techniques alone or in combination to resolve somatic dysfunction. Treatments are either "direct," in which the physician engages a patient's restricted plane of motion, or "indirect," in which the body is placed into a position of ease during treatment.[3] OMT requiring a therapeutic correcting force is called an "active" treatment when provided by the physician and "passive" when provided by the patient. The most common OMT modalities used are described below.

OSTEOPATHIC MANIPULATIVE TREATMENT TECHNIQUES (TABLE 1)

High velocity low amplitude (HVLA). The HVLA technique is a direct, active technique in which the physician engages the pathological barrier of a joint restricted in a normal plane of movement.[3,4] Once a restricted "barrier" is engaged, the physician employs a quick thrust of short distance through the inhibited plane of motion.[4,5] The purpose of HVLA is to re-establish normal range of motion by directly engaging the restrictive component of a joint's somatic dysfunction. HVLA is contraindicated especially in patients with cervical rheumatoid arthritis, carotid or vertebrobasilar disease, bony metastasis, and severe osteopenia.[5]

Muscle energy. One cause of somatic dysfunction is postulated to derive from chronically contracted restrictor muscles affecting the body's normal range of movement.[2] Muscle energy is a direct, active treatment with broad

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TABLE 1. Summary of osteopathic manipulative therapy techniques

OSTEOPATHIC MANIPULATIVE TREATMENT	TYPE	MECHANISM OF ACTION	GENERAL INDICATIONS	DERMATOLOGICAL INDICATIONS	CONTRAINDICATIONS
High velocity low amplitude	Direct, passive	Physician engages plane of arthroidal restriction thrusting through dysfunctional joint with short, quick force	Somatic dysfunction affecting arthroidal structures of body	Burning mouth syndrome, notalgia paresthetica, brachioradialis pruritis	Cervical rheumatoid arthritis, carotid or vertebrobasilar disease, bony metastasis, severe osteopenia
Muscle energy	Direct, active	Physician moves patient to barrier of restriction and applies isometric counterforce as patient moves to neutral position for 3–5 seconds	Any area of the body with decreased range of motion	Burning mouth syndrome, notalgia paresthetica, brachioradialis pruritis	Patients with low vitality, fractures, unstable joints, recent surgery
Counterstrain	Indirect, passive	Patient is placed into position of ease that relieves pain sensation of tender point by 70% and held for 90 seconds	Muscle tenderness due to a distinct and predictable tender point	Burning mouth syndrome, notalgia paresthetica, brachioradialis pruritis, vulvodynia, analdynia, scrotodynia	Fractures, torn ligaments or tendons
Myofascial release	Direct/indirect, passive	Restricted fascia is encouraged into ease by palpatory guidance of physician	Any somatic dysfunction of the body with myofascial involvement	Burning mouth syndrome, notalgia paresthetica, brachioradialis pruritis, vulvodynia, analdynia, scrotodynia, hyperhidrosis	Open tissue wounds, fractures, recent surgery, deep vein thrombosis, neoplasms, internal injury
Craniosacral	Direct/indirect, passive	Manipulation of cranium in order to normalize physiological motion of cranial bones, dural membranes, and cerebrospinal fluid	Somatic dysfunction affecting the primary respiratory mechanism, articulatory, and myofascial components of cranium	Burning mouth syndrome, trigeminal neuralgia, Sjogren's syndrome, hyperhidrosis	Lack of biomechanical dysfunction, recent trauma, unwillingness to receive treatment
Lymphatic	Direct/indirect, passive	Varying techniques which promote normal drainage of fluid from congested tissues	Stasis of lymph or venous blood	Lipodermatosclerosis, elephantiasis nostras verrucosa, stasis dermatitis	Metastatic cancer, certain infections (e.g., tuberculosis), hypercoagulable states

applications to any part of the body restricted in motion. In treatment, the physician engages the restrictive barrier and asks the patient to voluntarily move from a precisely controlled position.[3] During the patient's effort, the physician provides an isometric counterforce for 3 to 5 seconds and then allows the muscle to relax for 3 to 5 seconds. A new restrictive barrier is then engaged and the process is repeated 2 to 4 times.[2] Muscle energy increases the length of hypertonic restrictor muscles and allows restoration of normal physiological motion.[2] The mechanism of action of muscle energy is thought to be related to the reciprocal inhibition of agonist/antagonist muscles and the Golgi tendon reflex.[6,7] When a stretch reflex engages an agonist muscle, its antagonist relaxes via reciprocal inhibition. Similarly, as sufficient tension is placed on the Golgi tendon organ of a muscle, reflex relaxation occurs in the previously hypertonic muscle. Muscle energy is contraindicated in patients with low vitality, fractures, unstable joints, and recent surgery.[8]

Counterstrain. Treatment using counterstrain is directed at discrete areas of tender tissue called tender points. Tender points are painful to the touch and occur in

predictable locations throughout the musculature of the body. They are theorized to arise from an antagonist muscle in a state of "panic," lengthening in response to a strained and painful agonist.[9] Counterstrain is an indirect, passive technique in which the patient is positioned away from a restrictive barrier of motion. The physician monitors the tender point in one hand and holds the patient in a position of ease with the other for 90 seconds.[5] By placing the body into a position of maximal ease and comfort, the somatic dysfunction of the strained muscle should begin to resolve. Treatment is repeated until a 70-percent reduction in tenderness is reported by the patient.[3] Tender points can be found in acute and chronic conditions and may be the primary indicator of somatic dysfunction or appear secondary to another medical cause.[9] Absolute contraindications for counterstrain occur in patients with fractures or torn tendons in areas of dysfunction.[9]

Myofascial release. Myofascial release (MFR) is a technique that focuses on fascia and the surrounding muscles.[3] Having found an area of myofascial strain, the physician applies compression or distraction forces to the somatic dysfunction using palpatory feedback to guide the strain to resolution.[10] MFR techniques either directly or indirectly engage restrictive barriers depending on the physician's perceived response of the fascia to palpation. The effectiveness of myofascial techniques is explained via the concept of tensegrity.[10] A tensegrity structure consists of multiple, non-touching rods balanced by a continuous tension system. If one component fails, so does the entire structure. Applying this concept to the human body suggests that bones are the rods and the continuous tension system is the myofascial and ligamentous tissues of the body. Therefore, myofascial strain theoretically has influences across the entire body and resolution allows return of a more balanced homeostatic equilibrium. The use of MFR depends on safe introduction of motion upon dysfunctional tissue. Consequently, MFR is contraindicated for patients with open wounds, fractures, recent surgery, deep vein thromboses, an underlying neoplasm, or other internal injuries.[10]

Craniosacral. Osteopathy in the cranial field was pioneered by a student of A.T. Still's named William Sutherland, DO, (1873–1954)[3] and remains one of the more controversial areas of osteopathic manipulative medicine.[11] Central to the cranial technique is the concept of the primary respiratory mechanism, which consists of the inherent rhythmic motion of the brain and spinal cord, fluctuation of cerebrospinal fluid (CSF), mobility of intracranial and intraspinal membranes, the articular mobility of cranial bones, and the involuntary mobility of the sacrum between ilia.[12] The goals of various treatment modalities are to normalize nerve function, eliminate circulatory stasis, normalize CSF fluctuation, release membranous tension, correct cranial articular strains, and modify gross structural patterns.[12] It is important to note that many treatment modalities across the spectrum of OMT are applied to the cranium in addition to those affecting the primary respiratory mechanism. Cranial technique requires special training and should be performed only by certified practitioners. Cranial manipulation is contraindicated for patients with recent trauma, a lack of biomechanical dysfunction or an aversion to receiving treatment.[13]

Lymphatic treatment. Most OMT treatments have an effect on lymphatic circulation.[14] As somatic dysfunction resolves, the body's natural homeostatic mechanisms are normalized and lymphatic drainage is naturally enhanced. However, lymphatic treatments remove impediments to lymphatic flow and augment the clearance of lymph and other immune elements from specific congested tissues. All lymphatic techniques begin with treatment of somatic dysfunction in areas known as "choke points." The three choke points—the thoracic inlet, respiratory diaphragm, and pelvic diaphragm—are areas that can impede lymph between body compartments when dysfunctional.[15] Choke points are treated with MFR assisted by respiration. Once obstruction is reduced, lymphatic treatments employ pumping, soft tissue, and manual drainage techniques to promote fluid movement. Lymphatic techniques are contraindicated in the presence of metastatic cancer, certain infections (e.g., tuberculosis), and hypercoagulable states.[5]

DERMATOLOGICAL DISEASES AND APPLICATION OF OSTEOPATHIC MANIPULATIVE TREATMENT (TABLE 2)

Dysesthesia syndromes. *Burning mouth syndrome (BMS).* BMS is a chronic pain syndrome characterized by burning or stinging feelings affecting the oral mucosa in the absence of clinically detectable signs.[16] BMS usually affects middle-aged women[17–20] and commonly presents with a "symptomatic triad" of chronic, unremitting pain, dysgeusia, and xerostomia.[21] Current pharmacotherapy consists mostly of antidepressants, antipsychotics, antiepileptics, and analgesics.[16] Due to the unwanted side effects of pharmacotherapy, OMT may serve as supplemental treatment for BMS.

Research in the last decade has suggested there is an underlying autonomic nerve disorder of the oral cavity in patients with BMS due to dysfunction of the sensory trigeminal nervous system.[21] This evidence is supported by the presence of neuropathic symptoms, including pain, dysgeusia, and xerostomia. It has also been suggested that BMS results from a reduction in salivary output.[18,22,23] if the etiology of BMS were proven to be of trigeminal nerve origin, cranial osteopathic manipulation to normalize neural function might be beneficial. This would require more definitive research into the etiology of BMS itself as well as the true effects of cranial manipulation, which have so far remained elusive in well-controlled clinical research trials.

The presence of xerostomia in BMS also suggests the disease may involve hypofunctioning of the parasympathetic nervous system (PNS). Many OMT treatments target the PNS in order to normalize its activity. For example, sphenopalatine ganglion release is a technique performed using the fifth finger to manually massage and stimulate the sphenopalatine ganglion located

TABLE 2. Summary of dermatological diseases and application of osteopathic manipulative therapy (OMT)

DERMATOLOGICAL DISEASE	CLINICAL FEATURES	PATHOPHYSIOLOGY	OSTEOPATHIC MANIPULATIVE THERAPY	RATIONALE
Burning mouth syndrome	Burning pain, dysguesia, xerostomia, muscle pain in face, neck, and shoulders	Possible autonomic dysfunction of trigeminal nerve	Craniosacral, muscle energy, MFR, counterstrain, HVLA	Craniosacral OMT to normalize parasympathetic nervous system and xerostomia. Other OMT to reduce associated muscle tenderness and pain
Notalgia paresthetica	Midscapular itch, macular amyloidosis, pain, hyperesthesia, and paresthesia	Unknown but possible thoracic nerve impingement and anatomical susceptibility	Muscle energy, myofascial release, high velocity low amplitude, counterstrain	OMT treats muscles surrounding thoracic nerve roots to reduce symptoms of impingement
Brachioradialis pruritis	Solar dermopathy, xerosis, pruritis on outer surface of arm	Associated with chronic sun exposure, cervical ribs and cervical nerve root impingement	Muscle energy, myofascial release, high velocity low amplitude, counterstrain	OMT treats muscles surrounding cervical nerve roots to reduce symptoms of impingement
Trigeminal neuralgia	Sudden, recurrent stabbing pain in distribution of trigeminal nerve branch(es)	Potentially due to demyelination of trigeminal nerve sensory fibers or compression of trigeminal nerve root entry zones	Craniosacral	Craniosacral OMT may be beneficial to treat somatic dysfunction of cranial bones and dural membranes reducing stress on trigeminal nerve root entry zones
Vulvodynia	Burning vulvar discomfort, increased pelvic floor muscle tonicity	Possibly due to nerve compression and/or myofascial hypertonicity	Counterstrain, myofascial releas	Reduce tenderness of pelvic floor muscles and compression of pudendal nerve roots
Lipodermatosclerosis	Lower extremity panniculitis, hyperpigmentation, ulceration, and sclerosis	Static blood in lobular capillaries leads to pannicular ischemia, necrosis, and fibrosis via unclear mechanism	Lymphatic treatments	Remove blockage to venous flow and balance fluid regulatory mechanisms
Elephantiasis nostras verrucosa	Hyperkeratosis, papillomatosis, verrucous lesions of the lower extremity	Due to chronic non-filiaral lymphedema and associated inflammatory response	Lymphatic treatments	Remove blockage to lymphatic flow and balance fluid regulatory mechanisms
Stasis dermatitis	Hyperpigmentation, hyperkeratosis, itching, and ulceration of lower extremity	Caused by blood leaking out of veins secondary to venous stasis	Lymphatic treatments	Remove blockage to venous flow and balance fluid regulatory mechanisms
Hyperhidrosis	Excessive sweating primarily affecting palms, axillae, and soles	Heritable hyperfunctioning of sudomotor sweat control system	Craniosacral, rib raising	Normalize excessive sympathetic output to sudomotor sweat control system

in the superior, posterior lateral area of the pharynx. The sphenopalatine ganglion's parasympathetic innervation derives from the facial nerve and contains sensory fibers from the trigeminal nerve. Stimulation of the ganglion may reduce xerostomia and pain associated with BMS by normalizing the activity of the PNS and sensory components of the trigeminal nerve. Sphenopalatine ganglion release may also benefit patients with other causes of xerostomia, such as Sjogren's syndrome. The upper thoracic spine represents the sympathetic autonomic preganglionic origin of fibers that then synapse at the superior cervical ganglia and innervate the head and neck. Treating both the upper thoracic and cervical region will address dysfunction and its related nervous associations in those areas.

Patients with BMS also suffer from pain in the temporomandibular joint and muscles of the jaw, neck, shoulder, and suprahyoid areas.[24] Although it is unclear the relationship between these symptoms and mouth pain, OMT could be used to alleviate muscle tension and pain in these areas. Patients would likely benefit from muscle energy, MFR, or counterstrain treatments. HVLA may also be considered for restricted muscle groups with cervical spinal attachments (e.g., splenii, trapezius, erector spinae, and scalene muscles).

Notalgia paresthetica. Notalgia paresthetica is a sensory neuropathy involving the dorsal spinal nerves typically presenting with a unilateral midscapular itch.[25–28] The dermatological manifestation is macular amyloidosis, a well-circumscribed hyperpigmented patch believed to be secondary to chronic scratching.[29–31] Notalgia paresthetica is accompanied by episodic bouts of pain, hyperesthesia, and paresthesia.[30,32,33] Although the exact pathophysiology of notalgia paresthetica is not understood, Massey and Pleet[34] proposed that the spinal nerves of vertebrae T2 to T6 emerge from the multifidi muscles at right angles leaving them susceptible to chronic injury. More recently, a study by Savk et al[35] examined the spinal pathology of 61 lesions in 43 patients and found vertebral pathology in 60.7 percent of relevant lesions. These studies suggest the pathogenesis of notalgia paresthetica may involve a combination of spinal nerve impingement and anatomical susceptibility to injury.

If the etiology of nostalgia paresthetica is related to chronic injury of spinal nerves and vertebrae, then OMT should focus on reducing stress in these key areas. If treated properly, symptoms of pain, pruritis, and muscle tenderness may be reduced. One case report has been published on the effectiveness of OMT in treating nostalgia paresthetica using muscle energy, MFR, and other soft tissue techniques.[27] One particular MFR technique, occipital release, was used to release fascial tension around the occiput. Occipital release is also thought to normalize PNS output due to its close proximity to the vagus nerve. Additionally, MFR was performed on the scapulothoracic fascia and muscle energy to the cervical and thoracic regions to relieve hypertonic local musculature. In practice, specific treatments are rationalized based on a patient's individual somatic dysfunction, but it is reasonable to assume the same OMT modalities apply to similar pathology.

HVLA and counterstrain may also be considered in the treatment of nostalgia paresthetica. Although HVLA is contraindicated in pathological vertebral segments, it may relieve somatic dysfunction in more distant segments of the spine. One case report has shown that exercises that strengthen rhomboid and latissimus dorsi muscles and stretch the pectoralis major can relieve symptoms of pruritis in notalgia paresthetica.[28] These trials further demonstrate the need for additional studies examining OMT's use in the treatment of nostalgia paresthetica.

Brachioradialis pruritis. Patients with brachioradialis pruritis (BP) develop unrelenting pruritis over the outer surface of the arm, elbow, and forearm with clinical evidence of xerosis and skin damage.[36] Contributing factors include chronic sun exposure, cervical rib, and cervical nerve root compression.[37,38] Authors agree that BP is a solar dermopathy in which ultraviolet (UV) light exposure triggers spinal dysfunction and pruritis.[39–41] Current therapy employs the use of nonsteroidal anti-inflammatory drugs (NSAIDs), topical capsaicin, gabapentin, physical therapy, and acupuncture.[42] The similar etiology of BP and NP suggests that the same OMT techniques used for the treatment of NP also apply to this BP. As mentioned above, treatments that relieve stress on the compressed cervical vertebrae include MFR, muscle energy, HVLA to the thoracic region, and counterstrain.

Trigeminal neuralgia. The International Association for the Study of Pain defines trigeminal neuralgia (TN) as "a sudden and usually unilateral severe, brief, stabbing, recurrent pain in the distribution of one or more branches of the fifth cranial nerve."[43] TN is uncommon with an estimated incidence of 4.5 cases per 100,000.[44] Women are affected twice as frequently as men typically between 50 and 60 years of age.[44] TN is also known as *tic douloureux* due to the abrupt onset of intense pain and muscle spasms during attacks that often cause patients to recoil. The sensation of pain commonly radiates to areas within the distribution of cranial nerve V2 and/or cranial nerve V3.[45] TN occurs in bouts lasting weeks to months, but symptom-free intervals shorten as the disease progresses.[46] Quality of life can severely deteriorate with TN and many patients develop secondary psychological problems.[47]

Several theories explain the pathogenesis of TN. The leading theory attributes the symptoms of TN to demyelination of trigeminal sensory fibers within the proximal nerve root.[48] This theory is supported by the presentation of TN symptoms in patients with multiple sclerosis. Another theory suggests that compression of the trigeminal nerve at the root entry zone by a blood vessel contributes to the symptoms of TN.[49] There is evidence to support both theories, but no cause and effect relationship has been clearly defined.

Current first-line pharmacotherapy for TN is carbamazepine. Other medications include phenytoin, gabapentin, baclofen, and botulinum toxin A.[50] Patients refractory to medical therapy may investigate surgical

techniques including vascular decompression and gamma radiation.[46] OMT may be useful as an adjunct to primary therapy for symptomatic relief. As in BMS, overactivity of the trigeminal nerve could be normalized using sphenopalatine ganglion release. This technique may reduce the activity of the trigeminal nerve and decrease sensations of pain. From an anatomical standpoint, branches V1, V2, and V3 of the trigeminal nerve exit the cranium through the superior orbital fissure, foramen rotundum, and foramen ovale, respectively. Manipulation of the cranium and its bony articulations by an experienced osteopathic physician could relieve trigeminal nerve root compression where its branches exit the cranium. Treatment of the associated preganglionic regions of the thoracic spine and synaptic levels in the cervical spine along with treatment of the sphenopalatine ganglion may be helpful in normalizing the autonomic relationships to the region. In addition, treatment of the superior thoracic aperture and cranial base may be helpful in facilitating fluid motion. Trigger points in the temporalis muscle may be implicated in generating pain to the region of the trigeminal nerve and can be treated as well.[51]

Vulvodynia. Since 2003, vulvodynia has been defined as "vulvar discomfort, most often described as burning pain, occurring in the absence of relevant visible findings or a specific, clinically identifiable, neurological disorder."[52] Feelings of discomfort may also present with a combination of stinging, irritation, itching, pain, rawness, allodynia, hyperalgesia, and dysparenuria.[53,54] Vulvodynia is estimated to affect as many as 15 percent of all women.[55] The etiology is unknown, but the disease has been linked to developmental abnormalities, deoxyribonucleic acid (DNA) polymorphisms, peripheral and central neuropathic processes, nerve compression, myofascial hypertonicity, and an increased density of C-afferent nociceptive fibers in the vestibular mucosa.[56] Symptoms of allodynia and hyperalgesia suggest sensorineural dysfunction or central neuropathic pain processes.[57] Diseases like scrotodynia and analdynia are also neuropathic in origin; therefore, OMT considerations for vulvodynia may benefit these patients as well.

There are a variety of therapeutic options for vulvodynia, including topical anesthetics, oral medicines (e.g., antidepressants, gabapentin), clothing modifications, physical therapy, steroid injections, and surgery.[54,56] However, due to the multidimensional etiology of vulvodynia, no standard treatment protocol applies to every patient. Interestingly, a previous cohort study by Bergeron et al[58] surveyed 35 women and found that 71 percent reported complete, great, or moderate response to physical therapy. Several other studies have reported a 60- to 80-percent success rate in patients receiving biofeedback and physical therapy.[59] These findings support the use of OMT in the treatment of vulvodynia.

Many patients with vulvodynia have increased muscle tonicity, pain, and trigger points of the levator ani muscles of the pelvic diaphragm.[56] Pelvic diaphragm release, a form of MFR assisted by respiration, could be used to resolve hypertonicity of the pelvic floor, reduce pain, and restore local homeostatic regulatory mechanisms. Counterstrain may also be used to treat tenderpoints within the pelvic floor muscles. The pelvic floor muscles and vulva are innervated by the pudendal nerve from the S2 to S4 nerve roots. OMT cannot reverse arthritic changes, but a technique called spontaneous ligamentous release can liberate pudendal nerve entrapment in the sacroiliac area. This direct and indirect technique is a subtype of MFR applied to the ligaments attaching the sacrum and ilium. Naturally, patient education, consent, and comfort are of utmost importance before performing OMT on sensitive areas of the body.

LIPODERMATOSCLEROSIS, ELEPHANTIASIS NOSTRAS VERUCCOSA, AND STASIS DERMATITIS

Lipodermatosclerosis (LDS), also known as sclerosing panniculitis, is a form of lower extremity panniculitis typically affecting middle-to-late aged females.[60,61] Clinically, the acute inflammatory phase of LDS is characterized by painful, symmetric, red-to-purple, poorly demarcated, indurated plaques in a stocking-like distribution on the medial leg.[62,63] LDS develops a classic "inverted champagne bottle" appearance in the chronic phase characterized by diffuse fibrosis, sclerosis, and hyperpigmentation of the lower leg.[64] The pathogenesis of LDS begins with venous failure. Although the exact mechanism of these events remains unknown, static blood in lobular capillaries ultimately causes pannicular ischemia, fat necrosis, and fibrosis.

Two other dermatological conditions resulting from fluid stasis in the lower legs are difficult to treat. Elephantiasis nostras verrucosa is a rare condition associated with chronic nonfilarial lymphedema typically affecting the lower legs and feet.[65] Characteristic dermatological manifestations include hyperkeratosis, papillomatosis, and verrucous lesions in a bed of fibrosis secondary to lymphostasis and inflammation.[66–68] Stasis dermatitis is another disorder occurring as a result of venous stasis in the lower legs. Initially, blood and fluid leak out of congested veins causing hyperpigmented, pruritic lesions, which become hyperkeratotic and ulcerate over time.[69]

Current treatment options for stasis dermatoses include compression stockings, elevation, and diuretics. Additional treatments include stanozol and other anabolic steroids, such as oxandrolone and danazol.[63,64] Compression stockings are the standard treatment and help to reduce edema-induced ischemia, tissue damage, and ulceration.[63] Similarly, OMT that increases lymphatic flow and venous return may benefit patients with disease related to chronic stasis. As discussed previously, all lymphatic techniques must be preceded by normalization of relevant choke points, which may inhibit movement of lymph between body compartments. In the case of the lower extremity, applicable choke points are at the pelvic and thoracic diaphragm and require respiration-assisted MFR before lymphatic treatments.

Effleurage and pedal pump techniques are OMT

treatments used to increase lymphatic return from the lower extremity and decrease inflammation.[70] Effleurage is a manual soft tissue technique characterized by light or firm strokes of the palm and/or fingers on the tissue in a distal-to-proximal direction. This technique is meant to directly mobilize lymph and venous blood toward the superior end of the body and can be assisted by elevation of the limb. The pedal pump is another technique used to augment lymphatic return performed by light dorsiflexion and plantarflexion of the ankle at a rate of 120 cycles per minute while the patient lays supine.[70] This technique takes advantage of the fascial connections between the feet and upper body utilizing the movement of muscles to enhance mechanical influence on lymph mobilization. Finally, recent research has shown that the sclerosis typical of the chronic state of LDS is the result of accumulated hyalinized collagen fibrils with an abnormal number of cross links.[71] Therefore, manual stretching techniques of the sclerotic area may have an effect on reducing tension within the lesion by disrupting the collagen fibrils and their excessive cross links. Nonetheless, the role of OMT in the treatment of stasis dermatoses awaits further evaluation.

HYPERHIDROSIS

Hyperhidrosis is a common disorder affecting 0.6 to 1 percent of the Western population[72] characterized by excessive function of the sudomotor sweat control system typically affecting the palms, axillae, and soles.[73] Primary hyperhidrosis generally appears in adolescence and is believed to be inherited in an autosomal dominant fashion with variable penetrance.[74] In contrast, secondary hyperhidrosis is caused by other medical conditions and may present at any age. The exact pathophysiology of hyperhidrosis is unknown, yet a lack of excessive sweating during the night suggests a strong emotional component[75] in addition to localized hyperfunctioning of sympathetic fibers passing through the T2 and T3 ganglia.[76] Nonsurgical therapy employs topical aluminum chloride, iontophoresis, anticholinergics, botulinum toxin, and relaxation therapy.[73] The gold standard of surgical therapy is a thoracoscopic sympathectomy, a procedure that interrupts preganglionic sympathetic nerve impulses supplying sweat glands from the T2 to T4 dorsal chain ganglia.[73] Although the majority of patients show long-term resolution with surgery, most patients experience compensatory hyperhidrosis that can be severe in 1 to 2 percent of cases.[73]

Before considering surgical options, OMT may be a valuable supplement to pharmacotherapy. Sympathetic nerve fibers emerge from thoraco-lumbar vertebrae and their ganglia lie in the paravertebral region. Due to the proximity of sympathetic ganglion, OMT directed toward the vertebral bodies and ribs protruding from them may normalize sympathetic output at the level of the ganglion. An articulatory technique called rib raising calls for superior pressure applied to the inferior aspect of ribs at the level of a ganglion of interest.[77] By decreasing somatic dysfunction in the area of the sympathetic chain ganglion, the excess autonomic output of hyperhidrosis may become normalized. Occipital release is a technique described previously that augments the output of the PNS and may help restore balance within the autonomic nervous system. Finally, cranial manipulation may have effects on the output of sympathetic activity and must be investigated as a potentially beneficial treatment as well.

CONCLUSION

Although the dermatology office is a fast-paced environment that may not be conducive to OMT practice on a daily basis, several skin diseases may benefit from OMT. Dysesthesia syndromes, stasis dermatoses, and hyperhidrosis commonly present to dermatologists and are clinically challenging to treat due to their cutaneous, vascular, immune, neural, and psychological manifestations. Although some clinicians are not trained or familiar with the application of OMT, they should be aware of its therapeutic potential and consider a referral to an OMT specialist. As demonstrated in this article, dysesthesia syndromes, stasis dermatoses, and hyperhidrosis have underlying pathophysiologies that may improve with OMT. In some cases, OMT may even prevent the need for invasive procedures or drugs with an adverse side effect profile. Nonetheless, further studies are necessary to confirm the therapeutic benefit of OMT and its application to the treatment of dermatological disease.

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